The bHLH Repressor Deadpan Regulates the Self-renewal and Specification of Drosophila Larval Neural Stem Cells Independently of Notch.

Journal: PLoS One

Publication Year: 2012

Authors: Sijun Zhu, Jill Wildonger, Suzanne Barshow, Susan Younger, Yaling Huang, Tzumin Lee

PubMed link: 23056424

Funding Grants: Training Program in Stem Cell Research at UCSF

Public Summary:

Scientific Abstract:
Neural stem cells (NSCs) are able to self-renew while giving rise to neurons and glia that comprise a functional nervous system. However, how NSC self-renewal is maintained is not well understood. Using the Drosophila larval NSCs called neuroblasts (NBs) as a model, we demonstrate that the Hairy and Enhancer-of-Split (Hes) family protein Deadpan (Dpn) plays important roles in NB self-renewal and specification. The loss of Dpn leads to the premature loss of NBs and truncated NB lineages, a process likely mediated by the homeobox protein Prospero (Pros). Conversely, ectopic/over-expression of Dpn promotes ectopic self-renewing divisions and maintains NB self-renewal into adulthood. In type II NBs, which generate transit amplifying intermediate neural progenitors (INPs) like mammalian NSCs, the loss of Dpn results in ectopic expression of type I NB markers Asense (Ase) and Pros before these type II NBs are lost at early larval stages. Our results also show that knockdown of Notch leads to ectopic Ase expression in type II NBs and the premature loss of type II NBs. Significantly, dpn expression is unchanged in these transformed NBs. Furthermore, the loss of Dpn does not inhibit the over-proliferation of type II NBs and immature INPs caused by over-expression of activated Notch. Our data suggest that Dpn plays important roles in maintaining NB self-renewal and specification of type II NBs in larval brains and that Dpn and Notch function independently in regulating type II NB proliferation and specification.